Bandolier 132

Independent evidence-based thinking about health care

Not given to questioning too much

Does it seem to you that we live in a very strange world, beleaguered by the inexplicable? Do you find yourself berating the TV, newspaper, or even respectable medical journals? If so, join the club: you are not alone.

The problem involves media correspondents who always have to give two sides of an argument, even if one is bonkers, and the whole peer-review process of medical journals that seems to let far too much nonsense through. It all comes down to not questioning too much.

An example comes this month from the world of acupuncture. Yet another paper shows acupuncture to be no different from something that is vaguely similar to, but completely unlike, acupuncture (a control). This time we can work out that both are the same as doing nothing, yet the paper claims it as proof positive that acupuncture is a good thing.

When we have good evidence about alternative medicine it is a bit worrying. A look at Ayurvedic herbal remedies shows that a good proportion contain dangerously high levels of lead, mercury, or arsenic, enough to cause real problems. How long have we known this? Since the mid-70s, yet without any moves to regulation. You can buy them in many countries, completely unregulated, because they are dietary supplements. Makes you wonder about paying taxes for all those regulatory authorities, doesn't it?

Impertinent questions

Bronowski believed that asking an impertinent question put us on the way to a pertinent answer. Pertinent answers require straight thinking. A few good examples of straight thinking to balance the less than straight.

Perhaps the best is a systematic review of antidepressants for bipolar depression. One definition of evidence-based medicine is to apply the best evidence from systematic research to the treatment of individual patients, and this review is a near-perfect blend of evidence, clinical experience, and care. Excellent teaching material. Another good example is emerging evidence about impaired fasting glycaemia, its consequences, and what to do about it. Another, asking questions about how treatments work for our diverse peoples, has been worth waiting for.

It is the failure to ask questions, pertinent or impertinent, that makes us fulminate. It is one thing to be patient, but some poet or another cautioned about bewaring the fury of a patient man.

Impaired fasting glycaemia

In recent years the diagnostic cut-off value for fasting glucose has been reduced from 7.8 mmol/L to 7.1 mmol/L. A new category of intermediate impairment of glucose metabolism has been introduced, that of impaired fasting glycaemia, with a fasting glucose of 6.1 to 6.9 mmol/L, to define a state intermediate between diabetes and normal glucose metabolism. This poses some questions, about the risk of progressing to diabetes with impaired fasting glycaemia, and about the risk factors, and how to deal with the condition.

Progression to diabetes

A study from Helsinki provides an answer about the risk of progressing to diabetes with impaired fasting glycaemia [1]. In 1987 it recruited several thousand adults aged 45-64 years and measured fasting glucose, and glucose two hours after a 75 gram oral load. Impaired fasting plasma glucose was defined as 6.1 to 6.9 mmol/L, and impaired glucose tolerance was defined as a two-hour plasma glucose between 7.8 and 11 mmol/L.

Those free of diabetes at baseline (fasting plasma glucose below 7 mmol/L and/or two-hour glucose below 11 mmol/L) were followed for 10 years, and details of incident diabetes obtained from a computerised record linkage system.

Results

There were 2,593 participants free of diabetes at baseline who provided 26,700 years of follow up. Impaired glucose tolerance was more common than impaired fasting glucose, and only 1.5% of participants had both impaired fasting glucose and impaired glucose tolerance (Table 1).

The risk of progression to diabetes over 10 years was significantly higher with impaired fasting glucose (Figure 1) and with impaired glucose tolerance (Figure 2). The risk of

In this issue

Table 1: Risk of developing diabetes over 10 years at different levels of normal or impaired glucose metabolism in a Finnish population

Fasting plasma glucose	Oral glucose load	Percent of total	Diabetes (%)	10 year risk
Normal	Normal	82.1	2.0	1 in 50
Impaired	Normal	4.0	4.6	1 in 22
Normal	**Impaired**	12.4	10.8	1 in 9
Impaired	**Impaired**	1.5	49.9	1 in 2

Figure 1: Initial fasting plasma glucose and risk of diabetes over 10 years

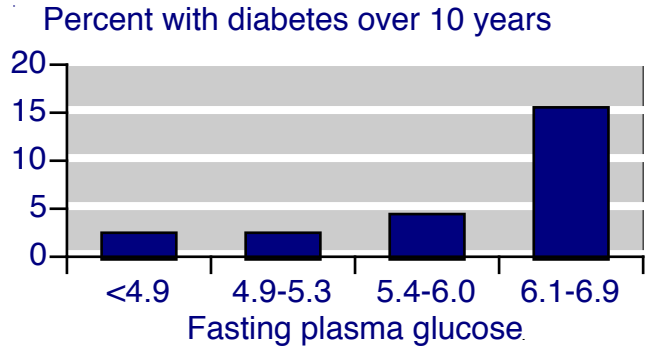
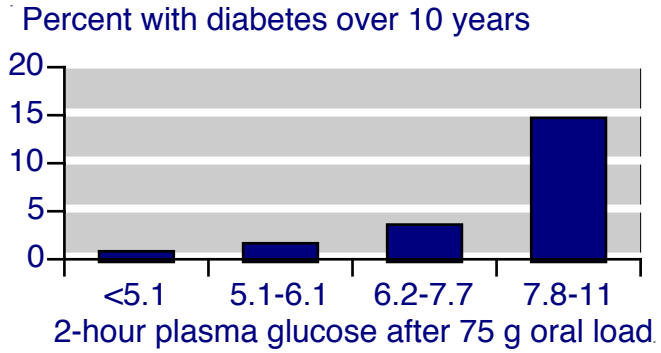


Figure 2: Plasma glucose 2 hours after 75 g load and risk of diabetes over 10 years



developing diabetes over 10 years was 1 in 50 for an adult aged 45-64 years with normal fasting glucose and glucose tolerance, and rose to 1 in 2 for an adult with both tests impaired. For impaired fasting glucose the risk was 1 in 22 (Table 1). Men and women had the same level of risk.

Risk factors

A Danish study [2] looked at risk factors in persons with impaired fasting glycaemia and those with impaired glucose tolerance. It surveyed 1,374 adults aged 20-69 from a single practice (66% of eligible patients), and under standardised conditions measured both fasting blood glucose and performed a glucose tolerance test with a 75 gram oral load. Physical examination and patient questionnaire gathered information on risk factors.

Results

Of the 1, 374 patients, 50 had diabetes according to the WHO (1999) criteria. Nineteen had been previously diagnosed, but 31 (2.3%) were previously not known to have diabetes. In all of them the diagnosis was confirmed on re-examination.

Fifty-one adults (3.7%) had impaired fasting glycaemia (defined here as fasting blood, as opposed to plasma, glucose of 5.6 to 6.0 mmol/L), and 90 (6.6%) had impaired glucose tolerance (two hour blood glucose of 6.7 to 9.9 mmol/L). There were no differences between these two groups for any of a whole range of risk factors for vascular disease. The mean age was about 50 years, about half were women, the average BMI was 28, and the average cholesterol 5.6 mmol/L.

Lifestyle intervention

A large US randomised trial [3] shows that lifestyle modification is an effective intervention for preventing or delaying progression from impaired fasting glycaemia to diabetes. The study recruited adults of 25 years or older with a BMI of 24 or higher and a fasting plasma glucose of 5.3 to 6.9 mmol/L or plasma glucose two hours after a 75 gram oral load of 7.8 to 11 mmol/L. They were randomised to placebo plus standard lifestyle recommendations in the form of written information and an annual session encouraging healthy lifestyle, or intensive lifestyle interventions aimed at weight reduction, exercise, and low-calorie, low-fat diet taught on a personal basis in 16 sessions over the initial 24 weeks. The outcome was progression to diabetes. There was also a metformin-treated group, but that is not reported here.

Results

In the three groups there were 3,234 participants, with 2,161 in placebo and intensive lifestyle groups. They were followed for an average of 2.8 years. Half the participants in the intensive lifestyle group had lost 7% or more of their body weight at 24 weeks, and 38% maintained their weight loss to the end of the study. The majority (60%) had maintained a weekly goal of 150 minutes of physical exercise a week by the end of the study. After one year, the average daily calorie reduction was 250 kcal in the placebo group and 450 kcal in the intensive lifestyle group.

Diabetes incidence was 58% lower in the intensive lifestyle group than the placebo group (Table 2), with a crude number needed to treat to prevent one case of diabetes over 2.8 years of 16. Metformin treatment without the intensive lifestyle intervention achieved a 31% reduction in incidence of diabetes.

Table 2: Development of diabetes over 2.8 years with placebo and lifestyle modification

	Percent with diabetes		
	Placebo	Lifestyle	Crude NNTp
Overall	11	4.8	16.1
Fasting plasma glucose (mmol/L)			
5.3-6.1	6.4	2.9	28.6
6.1-6.9	22.3	8.8	7.4
Two hours after 75 gram oral glucose load (mmol/L)			
7.8-8.5	7.1	1.8	18.9
8.5-9.6	10.3	4.4	17.0
9.6-11	16.1	8.5	13.2

Table 3: Percentage of adults in Scandinavia with different levels of glycaemic control

Fasting plasma glucose status	Glucose load status	Percent
Normal	Normal	84.6
Impaired	Normal	4.0
Normal	**Impaired**	10.2
Impaired	**Impaired**	1.3

The largest impact of intensive lifestyle intervention was in participants (about one third of the total) who had impaired fasting glycaemia with fasting plasma glucose of 6.1 to 6.9 mmol/L (Table 2).

Comment

Impaired fasting glycaemia is not uncommon. It affected about 4% of adults aged 20-64 in the two Scandinavian studies (Table 3). But of equal interest was that as many as 15% (1 in 8) adults had some form of impairment of glucose metabolism. Cardiovascular risk factors were about the same with whichever form of impaired glucose metabolism.

The bad news is that type 2 diabetes is much more likely to develop in people with impaired glucose metabolism, especially those with both impaired fasting glycaemia and impaired glucose tolerance, where the risk was 1 in 2 over 10 years. In the randomised trial [3] progression to diabetes in people with impaired fasting glycaemia was 22% over less than three years, while the Scandinavian study had only 5% over 10 years. The cause of this large discrepancy is not obvious. There was not such a big difference in participant characteristics, so perhaps it represents something of a cultural divide between the USA and Finland.

The good news is that people with impaired fasting glycaemia can change their lifestyle to stave off, or delay, the onset of diabetes. It's the same old story: lose weight, exercise, and eat a healthy diet. But the sting in the tail is that if progression to diabetes is lower in Europe than the USA, the effects of better lifestyle, while good for their own sake, may be less evident. Getting to the root of the large differences in progression to diabetes would be illuminating.

References:
1 Q Qiao et al. Progression to clinically diagnosed and treated diabetes from impaired glucose tolerance and impaired fasting glycaemia. Diabetes Medicine 2003 20: 1027-1033.
2 PE Heldgaard et al. Impaired fasting glycaemia resembles impaired glucose tolerance with regard to cardiovascular risk factors: population-based, cross-sectional study of risk factors for cardiovascular disease. Diabetes Medicine 2004 21: 363-370.
3 Diabetes Prevention Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. New England Journal of Medicine 2002 346: 393-403.

Exercise and sleep

Imagine going to sleep at 10 and waking at seven the next morning. For some that is an unachievable nirvana, because of nights of broken sleep, or no sleep. A good night's sleep, even occasionally, is something many would love to have.

Being tired would seem to be a useful factor in promoting good sleep, but it isn't always like that. Or perhaps it is the right sort of tired – the tired that comes from physical, rather than mental, exercise. Bandolier performed a quick search for studies that might shed some light on this. It found three [1-3], but they hold out only limited hope.

Older adults [1]

The eligibility criteria for this trial were extensive (12 items), but included an age range of 50-76 years, being sedentary, with a moderate sleep complaint (getting to sleep, waking during the night, or waking and getting up in the morning, and not taking sleep medicines. The intervention involved four exercise sessions a week for 16 weeks, two in a class and two at home, with each session lasting 60 minutes, half of which was endurance training.

Participants were randomised to exercise or no intervention control. Outcomes were standard sleep scales measured before, during, and after the intervention period, using sleep diaries and sleep quality indices.

Forty-eight people (average age 62 years) were randomised, with 20 completing the exercise programme and 23 completing the no intervention control period. Adherence to exercise was high. Initial sleep onset was about 27 minutes in exercise and control groups at baseline, and fell to 15 minutes after 16 weeks of exercise (Figure 1). Initial sleep duration was about six hours in exercise and control groups, and rose to 6.8 hours after 16 weeks of exercise (Figure 2). Better exercise duration was associated with better night-time sleep, and less daytime sleep.

Figure 1: Average time to fall asleep at the start and end of therapy for exercise group and no intervention control

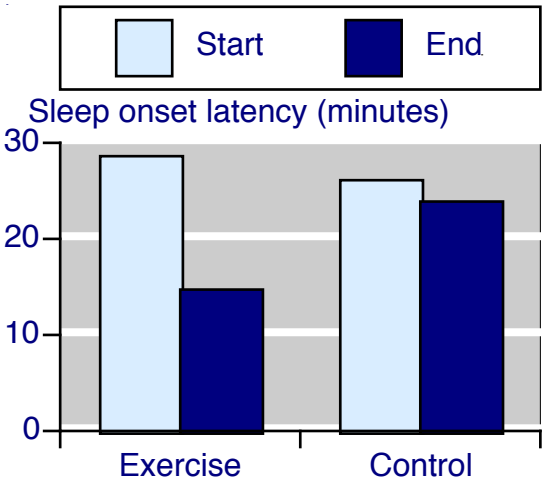
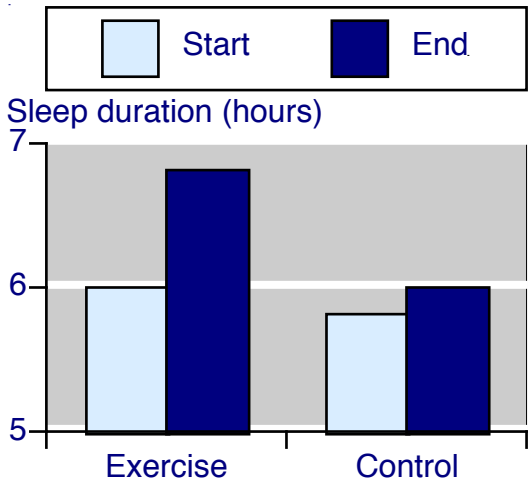


Figure 2: Average sleep duration at the start and end of therapy for exercise group and no intervention control



Depressed adults [2]

Eligibility for this study was based on a proper diagnosis of unipolar major or minor depression of dysthymia, and age over 60 years, as well as aerobic exercise more than twice a week. The intervention was high-intensity progressive resistance training three days a week for 10 weeks. The control was enrolment in a health education programme without exercise training. Outcomes were standard sleep scales measured before and after the intervention period, using sleep diaries and sleep quality indices.

Twenty-eight participants had an average age of 71 years, and adherence to the exercise programme was high. Exercise reduced the proportion of poor sleepers (Figure 3), and more exercise participants reported improved sleep, compared with controls (Table 1). Six of 15 (40%) depressed adults in the exercise group reported improved sleep, compared with none in the control group. Though numbers were small, this implies a number needed to treat of about 2.5 for one depressed adult to have improved sleep. Actually, it may be better than that, because two patients in the control group actually reported worse sleep.

Figure 3: Poor sleepers among depressed adults at start and end of intervention for exercise and control groups

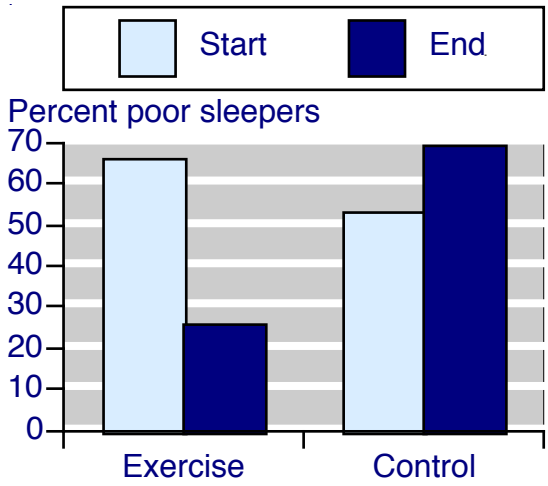


Table 1: Change in sleep quality at the end of the intervention for exercise and control groups

	Improved	Same	Worse
Exercise (n=15)	6	9	0
Control (n=13)	0	11	2

Postmenopausal women [3]

Eligible women were post-menopausal, taking no hormone replacement therapy. Smokers, and those with medical conditions where exercise was contraindicated were excluded. They were randomised to an exercise programme, or a stretching programme. Exercise involved moderate aerobic exercise five days a week for a year, some at a centre, and some at home. Stretching involved a 60-minute low intensity stretching and relaxation session each week for a year.

Over a year exercise or stretching made little difference to sleep quality indicators, though stretchers used less sleep medication. Because the study was designed to assess morning versus evening exercise, an analysis showed that morning exercisers who exercised for more than 225 minutes a week had significantly less trouble falling asleep, while evening exercisers who exercised more than 180 minutes a week had more trouble falling asleep.

Comment

This is not be the whole literature on exercise and sleep. There are other studies that may have included some exercise with other interventions, and there are certainly observational studies. Some of these also associate improved sleep with more exercise.

The down side is that there are not many trials of exercise for sleep. They are in people with different sources of problem (depressed, postmenopausal), and while they may be randomised none could be blind. They are small studies, with just over 200 participants.

Yet all show a consistent pattern of improved sleep measures with exercise. It may be that some threshold of exercise is needed to obtain better sleep, or perhaps morning exercise is better than evening exercise, but some measures of improved sleep were consistently found. Exercise is good for the heart, for weight, for lungs, and for the bones. Whatever the effect on sleep, it is going to do some good. So for those who have poor sleep and want to do something about it, a useful new year's resolution might be to get in at least four to six hours of moderate exercise a week.

References:

1 AC King et al. Moderate-intensity exercise and self-rated quality of sleep in older adults. A randomized controlled trial. JAMA 1997 277: 32-37.
2 NA Singh et al. A randomized controlled trial of the effect of exercise on sleep. Sleep 1997 20: 95-101.
3 SS Tworoger et al. Effects of a yearlong moderate-intensity exercise and a stretching intervention on sleep quality in postmenopausal women. Sleep 2003 26: 830-836.

Antidepressants for bipolar depression

The use of antidepressants as monotherapy for depression in patients with bipolar depression is one of those areas where there is genuine uncertainty about its value. There may be benefits, but there may also be harm. The benefit would be a reduction in depressive symptoms, though this is by no means certain. The harm may come from the induction of manic episodes or mood instability. A superb systematic review [1] helps in understanding the amount of evidence and the likely benefits and harms.

Systematic review

The review used an extensive search strategy for randomised double blind studies comparing antidepressants with placebo, or with other drug treatments. Many electronic databases were searched, with the use of a huge number of individual drug names, and without exclusion of language.

Patients included in the trials had to have a current depressive or mixed depressive/manic episode and with at least one previous episode of mania or hypomania. The main outcome measures were clinical response and remission rates derived from observer-rated symptom reductions, induction of mania or hypomania, and discontinuation.

Results

Twelve randomised, double blind trials were included, five with placebo comparators, four comparing antidepressants, and three comparing antidepressants to other types of drug. Most patients were in the placebo comparisons. Duration was 4-10 weeks, in adults, and with a majority of women.

Clinical response was found in 123/213 (58%) of patients on antidepressant, compared with 153/449 (38%) on placebo (Figure 1). Three of these studies, including the largest, had some or all patients on lithium or olanzapine. The relative benefit was 1.9 (95% confidence interval 1.5 to 2.3), and the number needed to treat to produce one patient with clinical response was 4.2 (3.2 to 6.4).

Switching to mania was rare, occurring in 11/287 (3.8%) of patients on antidepressant, compared with 23/492 (4.7%) on placebo (Figure 2). There was no significant difference, with a relative risk of 1.0 (0.5 to 2.0). Withdrawal was significantly lower in patients on antidepressant (32%) than on placebo (43%).

There is tentative evidence that tricyclic antidepressants may be less effective than other antidepressants, but this is based on a small number of trials with few patients. The same comparisons consistently found a higher rate of switching to mania with tricyclic antidepressants (19/184; 10%) than with other antidepressants (6/186; 3%), and though this was statistically significant with a relative risk of 2.9 (1.3 to 6.7), the number of events was very small.

Comment

There are some papers that it is both a joy and a privilege to read, because they combine a good analysis of the evidence with clinical wisdom, and simultaneously give a wider perspective to the subject. This is one such, and if it is not required reading in psychiatry (and outside psychiatry), then perhaps it should be, as it has all these qualities.

It beautifully dissects how small positive or negative studies have swayed opinion. For instance, it describes studies that have been included in other (non-systematic) reviews, despite having different diagnoses and no bipolar patients at all.

The bottom line is the conclusion, that, on the basis of current evidence, it is probably overcautious and potentially not in the best interests of patients to discourage the use of antidepressants for bipolar depression.

Reference:
1 HJ Gijsman et al. Antidepressants for bipolar depression: a systematic review of randomized, controlled trials. American Journal of Psychiatry 2004 161: 1537-1547.

Figure 1: Clinical response in randomised trials comparing antidepressant with placebo

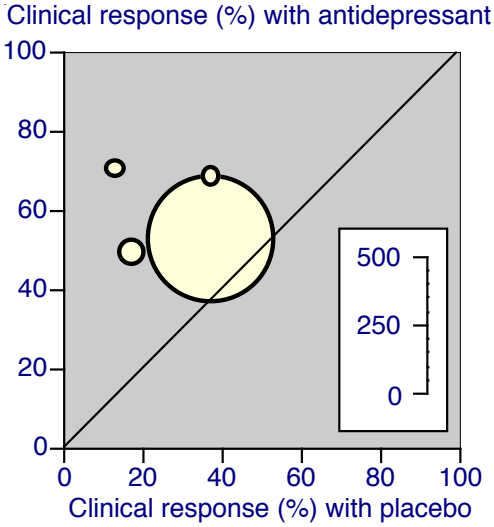
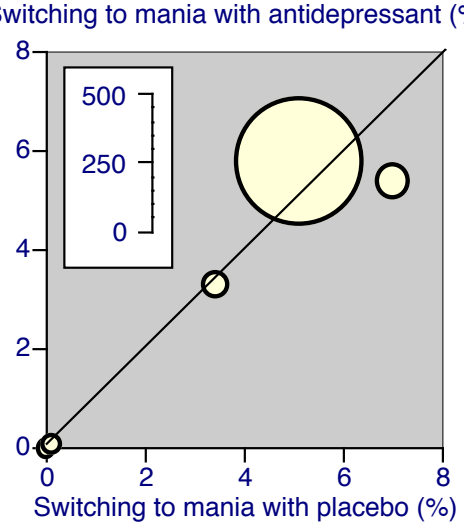


Figure 2: Switching to mania in randomised trials comparing antidepressant with placebo



Antihypertensive efficacy in black patients

Diversity is a wonderful thing, but awfully complicating when it comes to illness and therapy. So often terrific studies are performed, only to leave unanswered questions of whether results may differ by sex, or age, or race, or genomic makeup, let alone dose or intensity of the intervention, its duration, the severity of illness, or duration of disease. It is all very well hearing about yet another new gene being discovered that "may" help determine treatments sometime in the future, but that is pie in the sky tomorrow, and treating patients now is the name of the game, whatever their differences. We have some help with antihypertensive therapy in black patients, thanks to a detailed and enthusiastic systematic review [1].

Systematic review

This set out to identify all trials that looked at antihypertensive drugs in hypertensive black adults, using a series of electronic databases, including the Cochrane Library. There was no language restriction, and there were, in addition, considerable attempts to locate data by other means, including contacting authors for unpublished details.

Any randomised trial lasting at least two weeks was eligible if it had a placebo control and provided information on arterial blood pressure.

Results

Twenty-six trials were eligible, conducted in the USA, Caribbean, or Africa. Most trials (22) had quality scores that made bias unlikely. Most trials included patients with diastolic blood pressure in the range of 90-115 mmHg. The median duration of treatment was eight weeks.

Table 1 shows the main results, and with the exception of β-blockers for systolic blood pressure, all lowered blood pressure significantly more than placebo. The greatest blood pressure reduction, and largest proportion of patients achieving a diastolic blood pressure reduction goal, was with diuretics or calcium channel blockers. There was some suggestion that some β-blockers might increase systolic blood pressure. Overall only 23% of people in these trials achieved adequate diastolic blood pressure reduction.

Though an attempt was made to analyse the results according to different levels of initial blood pressure, limitations in the number of trials and patients, as well as different drugs and doses, made this something of a forlorn hope.

Adverse events were not analysed in detail, but headache, polyuria and nocturia, dizziness, tinnitus, bronchospasm, tachycardia, and cough were mentioned in some studies.

The review also sought mortality or morbidity outcomes in trials, and was able to calculate them for black patients in four trials. These trials all differed in patient characteristics, interventions, and endpoints. Some showed differences between black patients and white patients, and sometimes between black men and black women. Detailed reading of the review is needed to make the best sense of this for treating hypertensive black patients.

Comment

The good news is that the most commonly used antihypertensive drugs were effective in reducing blood pressure in hypertensive black people. It is probably essential reading for those writing guidelines. While there are always limitations in translating trials of antihypertensive monotherapy into clinical practice where, perhaps, multiple therapy is common, the addition of this evidence to clinical experience will be helpful, and add confidence to decisions made.

Reference:
1 LM Brewster et al. Systematic review: antihypertensive drug therapy in black patients. Annals of Internal Medicine 2004 141: 614-627.

Table 1: Evidence for efficacy of antihypertensive drugs in hypertensive black patients

Drug class	Number of		Mean BP reduction (mmHg more than placebo)		DBP goal reached (%)
	Trials	Patients	Systolic	Diastolic	
Diuretics	10	581	**11.8**	**8.1**	31
Calcium channel blockers	6	421	**11.6**	**7.8**	46
α-blockers	3	262	**7.4**	**3.4**	13
ACE inhibitors	7	451	**7.0**	**3.8**	10
Angiotensin II blockers	4	933	**3.6**	**3.4**	19
β-blockers	8	482	3.5	**5.4**	19

Statistically significant differences in bold, shaded cells; Number of trials and patients for the largest comparison (SBP or DBP)

Diastolic blood pressure (DBP) goal was ≤90 mmHg, or reduction of ≥10 mmHg, or 10% decrease

Ayurvedic herbal medicines and heavy metals

Bandolier just loves it when someone says that while alternative therapies may not have much evidence behind them, at least they do no harm. If only. Bandolier 104 reported that many herbal remedies "work" because they are adulterated with all sorts of drugs of low and high concentrations, and Chinese remedies for skin conditions commonly contain steroids. Bad things are known to happen because of this.

A study from Boston [1] examined Ayurvedic herbal remedies from south Asia and found that many contained high levels of the heavy metals lead, mercury, and arsenic.

Study

Researchers identified all the stores within 20 miles of Boston (Massachusetts, not Lincolnshire), and in mid-2003 visited the stores and bought one package of each Ayurvedic herbal remedy manufactured in south Asia and intended for oral use. No identical product was obtained more than once, but products with the same name but different manufacturer or different formulations were purchased. These were then analysed for lead, mercury, and arsenic.

Results

Seventy unique Ayurvedic products costing less than US$6 per package were obtained, manufactured in India or Pakistan, and carried in 30 stores. The most common indication for use was gastrointestinal disorders (70%).

Fourteen (20%) contained lead, mercury, and/or arsenic, some in very high concentrations. Figure 1 shows the concentrations found in micrograms (millionth of a gram, μg) per gram of herbal medicine. In Figure 1, the black horizontal bar represents the maximum recommended levels in some medicines in the USA, or the maximum chronic oral intake for mercury or arsenic, in micrograms. Concentrations of heavy metals in some herbal medicines were hundreds or thousands of times greater than this. Just to labour the point, please note that the concentrations in Figure 1 are on a logarithmic scale.

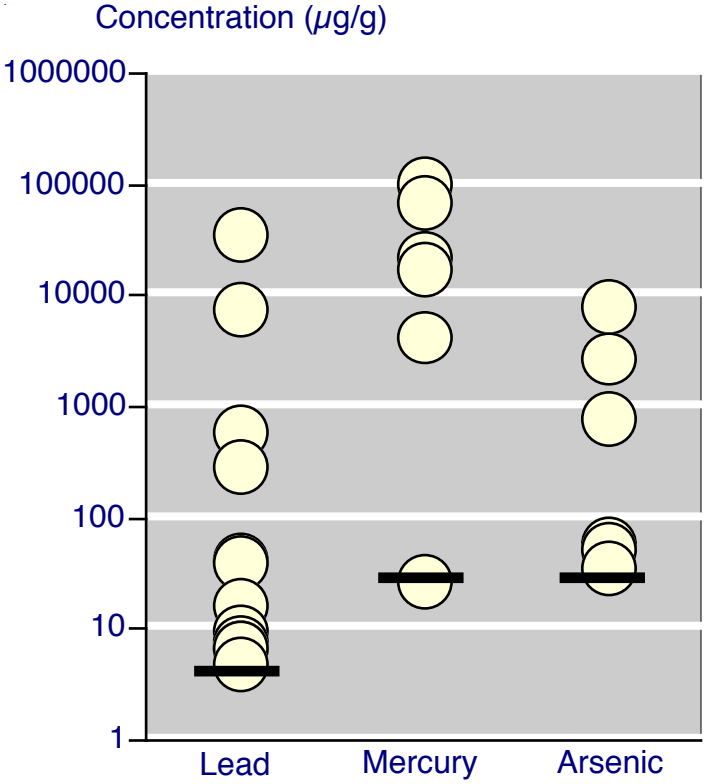
Duplicate samples were obtained for the herbal medicines with high heavy metal concentrations, and gave almost identical results.

As well as lead, mercury, and arsenic, some herbal medicines also contained high concentrations of tin or silver, but not gold or cadmium. Some of the medicines were for use in children.

Comment

This is not the first report of heavy metals in Ayurvedic medicines, and the paper reviews some of the rich literature. It tells us that the heavy metal concentrations in the medicines, if taken at recommended doses, would provide sufficient to be toxic and that serious toxicity has been reported.

Figure 1: Concentration (logarithmic scale) of lead, mercury, and arsenic in Ayurvedic medicines (μg/g) in those medicines where it was detected. The horizontal black bars approximate maximum recommended levels of oral intake



A review of traditional Indian remedies [2] shows that heavy metals, particularly lead, are regular constituents. This has repeatedly caused serious harm to patients taking such remedies. The incidence of heavy metal contamination was about 20% in this series [1], but the review found a study where 64% of samples collected in India contained significant amounts of lead (64% mercury, 41% arsenic and 9% cadmium). Both these papers have references to studies demonstrating heavy metal contamination of traditional Ayurvedic medicines. Ayurvedic principles apparently attribute important therapeutic roles to metals like mercury and lead, and these heavy metals have been the cause of significant illness.

There are two main lessons. The first is that knowledge that these medicines can contain high levels of heavy metals might make it worth asking patients with otherwise unexplained symptoms about their use. The other lesson is about testing and regulation. In a world that is so risk averse, how is it that people are allowed to purvey poisons to our fellows? The widespread notion of herbal medicinal products being inherently safe is at best naive and at worst downright dangerous.

Reference:
1 RB Saper et al. Heavy metal content of Ayurvedic herbal medicine products. JAMA 2004 292: 2868-2873.
2 E Ernst. Heavy metals in traditional Indian remedies. European Journal of Clinical Pharmacology 2002 57: 891-896.

Acupuncture for menopausal hot flushes

Evidence-based thinking occasionally means having to rub a few neurones together to make sense of something one reads. A perfectly good randomised trial of acupuncture, sham acupuncture, and oestradiol in menopausal women with hot flushes [1] is a useful test. It concluded that acupuncture and sham acupuncture work, but less well than oestradiol, and that acupuncture is a viable alternative, but not sham acupuncture. Is that a sensible conclusion based on the evidence?

Study

The report [1] presents data on three arms (acupuncture, sham acupuncture, oestradiol) of a five-arm study. There were 43 women in the three arms reported, who underwent two weeks of baseline data collection, and then 12 weeks of treatment, with later follow up which can be ignored, mainly because patient withdrawal reduces its value.

Acupuncture or sham acupuncture was given twice a week for two weeks, followed by once a week for 10 weeks. Sham acupuncture involved superficial needle insertion in the skin, one to five centimetres away from acupuncture points. Acupuncture involved electrostimulation and deeper insertion at true acupuncture points.

Results

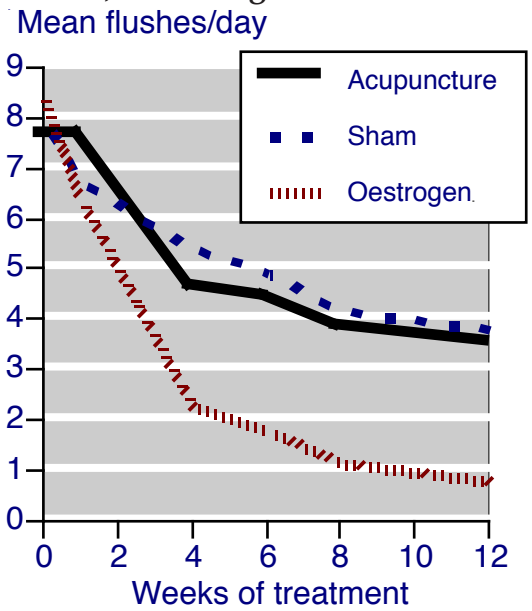
The number of hot flushes over 12 weeks of treatment in the three groups is shown in Figure 1. There was no difference between acupuncture and sham acupuncture over 12 weeks, but oestrogen reduced flushes to below one per day on average.

Comment

The obvious conclusion is that acupuncture doesn't work. Another conclusion, that drawn in the paper, is that this proves that acupuncture works by reducing daily flushes by half. We are treated to a long discussion that ambles through endorphins, effects of needles, is nothing if not comprehensive, as is the huge weight of statistical analysis used. But the headline conclusion found in the abstract (all that most will read) is that "acupuncture is a viable alternative treatment".

This is the problem we often find, that the "non-active, active" control works as well as the active. Here, though, we can shine a little more light on this problem. Table 1 shows the start and end of trial daily flush rate in this randomised trial, and the results for oestrogen and placebo found in a Cochrane review of the effect of oestrogens [2].

Figure 1: Average daily flushes in postmenopausal women treated with acupuncture, sham acupuncture, or oestrogen



It shows that the effects of oestrogen in the trial and review are about the same. It also shows that the effects of acupuncture or sham acupuncture are about the same as placebo. In other words, acupuncture is about as good as doing nothing, but it is much, much, more expensive.

When will alternative therapies really prove they work? While we wait will they stop fleecing people of huge amounts of cash for doing nothing?

References:

1 Y Wyon et al. A comparison of acupuncture and oral estradiol treatment of vasomotor symptoms in postmenopausal women. Climacteric 2004 7: 153-164.
2 AH MacLennan et al. Oral oestrogen and combined oestrogen/progestogen therapy versus placebo for hot flushes. The Cochrane Database of Systematic Reviews 2004, Issue 4.

Table 1: Average daily flushes in postmenopausal women at the start and end of treatment, in the randomised trial and in a Cochrane review

	Acupuncture (RCT)	Sham acupuncture (RCT)	Placebo (Sys Rev)	Oestrogen (RCT)	Oestrogen (Sys Rev)
Start of treatment	7.6	8.1	9.3	8.4	5.9
End of treatment	3.5	3.8	3.9	0.8	1.4